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LETTER TO THE EDITOR



Cyanide poisoning and antidotes

Dear Editor,

We read with interest the review article by Reade *et al.* titled, 'Review article: Management of cyanide poisoning'.¹ The article was a succinct review of current treatment guidelines for cyanide treatment. We concur with several of the authors' recommendations that had not been clearly stated in other reviews or reports. In particular, we agree that in cyanide-induced cardiac arrest, patients should receive hydroxocobalamin preferentially and repeat dosing as needed. We also agree that based on adverse effects alone, hydroxocobalamin might be a better choice for cyanide toxicity; in addition, we note a comparative study has been reported comparing sodium nitrite with hydroxocobalamin.² Finally, we also concur that the guidelines by the UK, which do not recommend antidotal treatment, are not based on current published evidence and that antidotes are effective for cyanide toxicity.

However, we disagree on three areas. First, the authors report that studies comparing supportive care with use of antidote have not been conducted; however, we reported a comparative trial between supportive care and antidotal therapy and found that supportive care in a cardiac arrest model is 100% lethal.³ Second, the authors report that most studies used thiosulphate in combination with antidotes and that there is evidence that sodium thiosulphate is effective alone. However, we reported a clinically relevant, critically ill model of cyanide-induced hypotension demonstrating that sodium thiosulphate is not effective alone and had 100% mortality.⁴ Finally, we disagree with the authors that sodium thiosulphate should be used following failure to respond to hydroxocobalamin. We demonstrated that sodium thiosulphate does not improve efficacy when combined with hydroxocobalamin.⁴ In

addition, based on previous animal and human studies, if the patient does not respond to the vasopressor or antidotal effects of hydroxocobalamin, the patient is unlikely to receive benefit from sodium thiosulphate, a drug that might take up to 30 min to take effect and is poorly transported into the mitochondrial membrane, hydroxocobalamin's site of action.^{4,5}

Competing interests

None declared.

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Vikhyat S Bebartá

Medical Toxicology, Department of Emergency Medicine,
San Antonio Military Medical Center,
San Antonio, Texas, USA

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